1991. International Business Communications (IBC).
DT Conference
FS DCCP
LA UNAVAILABLE

FILE 'HOME' ENTERED AT 16:21:24 ON 02 APR 96

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=> fil medl,cancerlit; d his l223-; dup rem l231
FILE 'MEDLINE' ENTERED AT 16:31:30 ON 02 APR 96

FILE 'CANCERLIT' ENTERED AT 16:31:30 ON 02 APR 96



     (FILE 'HOME' ENTERED AT 16:21:24 ON 02 APR 96)

     FILE 'MEDLINE, CANCERLIT' ENTERED AT 16:29:47 ON 02 APR 96
L223          22 FILE MEDLINE
L224          10 FILE CANCERLIT
     TOTAL FOR ALL FILES
L225          32 S (ANTIBODIES, BISPECIFIC AND RECEPTORS, FC+NT)/CT
L226          20 FILE MEDLINE
L227           9 FILE CANCERLIT
     TOTAL FOR ALL FILES
L228          29 S L225 AND (ANTIGENS+NT)/CT
L229           5 FILE MEDLINE
L230           6 FILE CANCERLIT
     TOTAL FOR ALL FILES
L231          11 S L228

     FILE 'MEDLINE, CANCERLIT' ENTERED AT 16:31:30 ON 02 APR 96


PROCESSING COMPLETED FOR L231
L232           8 DUP REM L231 (3 DUPLICATES REMOVED)

=> d 1-8 bib abs; fil hom

L232 ANSWER 1 OF 8  MEDLINE                                  DUPLICATE 1
AN   94267430     MEDLINE
TI   Antibody-dependent cellular cytotoxicity and neutralization of human
     immunodeficiency virus type 1 by high affinity cross-linking of gp41
     to human macrophage Fc IgG receptor using bispecific antibody.
AU   Mabondzo A; Boussin F; Raoul H; Le Naour R; Gras G; Vaslin B;
     Bartholeyns J; Romet-Lemonne J L; Dormont D
CS   Laboratoire de Neuropathologie experimentale et Neurovirologie,
     CRSSA, DSV/DPTE, Commissariat `a l'Energie Atomique,
     Fontenay-aux-Roses, France..
SO   JOURNAL OF GENERAL VIROLOGY, (1994 Jun) 75 ( Pt 6) 1451-6.
     Journal code: I9B. ISSN: 0022-1317.
CY   ENGLAND: United Kingdom
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals; Cancer Journals
EM   9409
AB   Human monocytes/macrophages, which express Fc receptors for IgG are
     involved in human immunodeficiency virus type 1 (HIV-1) infection
     and pathogenesis. These receptors are known to mediate numerous
     immunological functions including cell-mediated killing and possibly
     targeting of HIV to the lysophagosome monocyte-derived macrophage
```

(MDM) entry route for virus neutralization. To study both activities in HIV-1 infection, MDM Fc gamma RI was specifically selected using bispecific antibody (Bs-Ab) containing whole human monoclonal antibody against gp41 and the Fab' fragment of murine anti-Fc gamma RI 22.2 antibody. Bs-Ab was found to mediate potent antibody-dependent cellular cytotoxicity and virus neutralization.

L232 ANSWER 2 OF 8 CANCERLIT
AN 94602997 CANCERLIT
TI Phase Ia/Ib trial of bispecific monoclonal antibody (BsAb) therapy (anti-Her-2/neu x anti-CD64) (MDX-210) for breast or ovarian cancers that over express Her-2/neu (Meeting abstract).
AU Valone F H; Kaufman P A; Fanger M W; Guyre P M; Memoli V
CS Dartmouth-Hitchcock Medical Center, Lebanon, NH 03756
SO Proc Annu Meet Am Assoc Cancer Res, (1994). Vol. 35, pp. A1311. ISSN: 0197-016X.
DT (MEETING ABSTRACT)
(CLINICAL TRIAL, PHASE I)
(CLINICAL TRIAL)
FS ICDB
LA English
EM 9411
AB MDX-210 is a Fab' x Fab' BsAb constructed by chemically crosslinking Fab' mAb 520C9 (anti-Her-2/neu) and Fab' mAb22 (anti-CD64). CD64 is a high affinity Fc receptor for IgG. In vitro, MDX-210 effectively directs cytotoxicity and phagocytosis by monocyte-derived macrophages and IFNgamma-treated neutrophils. MDX-210 triggers release of TNFalpha by macrophages in the presence, but not in the absence, of HER-2/neu expressing target cells. The clinical trial's endpoints are evaluation of toxicity, determination of the MTD, and biological efficacy. Patients (pts) with stage IV breast cancer or stage III or IV ovarian cancer that is resistant to standard therapy and expresses Her-2/neu are eligible for treatment. Eight pts have been treated. Dose levels tested are 0.35, 1.0 and 3.5 mg/m2 given IV once at 6 mg/hr. Treatment was well tolerated. The principal toxicities were transient grade 1-2 fevers and malaise in 5 pts and grade 1-2 hypotension in 2 pts. MDX-210 was biologically active at all doses. Substantial, peripheral blood monocytopenia occurred in all pts by 1 hr and resolved by 24 hr. Mild neutropenia occurred at 1 hr and resolved by 2 hr. There were no changes in platelets or erythrocytes. Plasma TNFalpha increased to as high as 500 pg/ml in 5 of the 6 pts tested within 1-8 hr and resolved by 24 hr. Dose-dependent in vivo binding of MDX-210 to CD64 was observed with up to 80% saturation at 1 hr and significant binding for greater than or equal to 24 hr. No changes in T cell, B cell or NK cell subsets were observed 4 or 24 hr after treatment. MDX-210 is immunologically active at non-toxic doses and is a candidate for immunotherapy of Her-2/neu expressing tumors.

L232 ANSWER 3 OF 8 CANCERLIT
AN 94602995 CANCERLIT
TI Phase I trial of a bispecific murine monoclonal antibody targeting c-erbB-2 and CD16 (Meeting abstract).

AU Weiner L M; Ring D I; Li W; Palazzo I E; Davey M; Rivera V; Alpaugh R K
CS Fox Chase Cancer Center, Philadelphia, PA 19111
SO Proc Annu Meet Am Assoc Cancer Res, (1994). Vol. 35, pp. A1309. ISSN: 0197-016X.
DT (MEETING ABSTRACT)
(CLINICAL TRIAL, PHASE I)
(CLINICAL TRIAL)
FS ICDB
LA English
EM 9411
AB Bispecific monoclonal antibodies (BsMAb) can direct tumor lysis by effector cells via defined cytotoxic trigger molecules. The BsMAb 2B1 promotes c-erbB-2 tumor cell lysis by human NK cells and macrophages expressing CD16 (ie, FcgammaRIII), and is effective in murine xenograft models. Neutrophils (PMN) express an isoform of CD16 that does not trigger lysis. In a dose-escalating Phase I clinical trial, nine patients with c-erbB-2(+) tumors have been treated iv with 1 hr BsMAb infusions on days 1, 4, 5, 6, 7 and 8, at 1 mg/m2 (n=3) or 2.5 mg/m2 (n=6) 2B1 per dose. The MTD has not been reached. Treatment causes fevers, rigors, reversible neutropenia and loss of circulating monocytes and NK cells. Treatment also alters the distribution of 111In-labeled autologous leukocytes, with tumor localization noted in at least one patient. Circulating 2B1 retains its dual binding characteristics. Peak levels of 240-2260 ng/ml murine IgG have been detected, with binding to circulating and peritoneal PMN, NK cells and mononuclear phagocytes. One clinical response has been observed in a patient with chest wall recurrence of breast cancer. The binding of this BsMAb to CD16-expressing leukocytes has potent biological effects which may be exploited at higher, tumor-binding 2B1 doses.

L232 ANSWER 4 OF 8 CANCERLIT
AN 94600974 CANCERLIT
TI Phase I trial of 2B1, a bispecific murine monoclonal antibody targeting c-erbB-2 and CD16 (Meeting abstract).
AU Weiner L M; Ring D; Li W; Palazzo I E; Davey M; Rivera V; Alpaugh R K
CS Fox Chase Cancer Center, Philadelphia, PA 19111
SO Proc Annu Meet Am Soc Clin Oncol, (1994). Vol. 13, pp. A978. ISSN: 0736-7589.
DT (MEETING ABSTRACT)
(CLINICAL TRIAL, PHASE I)
(CLINICAL TRIAL)
FS ICDB
LA English
EM 9409
AB Bispecific monoclonal antibodies (BsMAb) can direct tumor lysis by effector cells via defined cytotoxic trigger molecules without competition by host IgG. The BsMAb 2B 1 promotes c-erbB-2 tumor cell lysis by human NK cells and macrophages expressing CD16 (ie, Fc gamma RIII), and is effective in a murine xenograft model. Neutrophils (PMN) abundantly express an isoform of CD16 that does

not trigger tumor lysis. In a dose-escalating Phase I clinical trial, eleven patients with c-erbB-2(+) tumors and ECOG PS = 0 or 1 have been treated iv with 1 hr BsMAb infusions on days 1, 4, 5, 6, 7 and 8, at 1 mg/m2 (n=3), 2.5 mg/m2 (n=6) or 5.0 mg/m2 (n=2) 2B1 per dose. The MTD has not been reached. The severity of fevers, rigors, transient Grade 4 neutropenia and the loss of circulating monocytes and NK cells are not dose-dependent. Peak serum levels of TNF (2400 pg/ml), IL-6 (9000 pg/ml), IL-8 (11 pg/ml) and elastase (600 ng/ml) are found 1-4 hr following the start of BsMAb infusion. Tumor localization of 111In-labeled autologous leukocytes has been noted. Dose-related peak serum levels of 240-2260 ng/ml murine IgG have been detected, as has HAMA. Circulating 2B1 retains its dual binding characteristics and can be found on circulating and peritoneal PMN, NK cells and mononuclear phagocytes by flow cytometry. Treatment enhances peripheral blood NK activity and LAK precursors, resulting in augmented in vitro growth inhibition of c-erbB-2(+) SK-OV-3 cells. A clinical response was observed in a patient with an evaluable chest wall recurrence of breast cancer. This first clinical trial of a BsMAb targeting tumor and CD16 has demonstrated potent biological effects which may be exploited at higher, tumor-binding 2B1 doses. The role of PMN in promoting these effects requires examination.

L232 ANSWER 5 OF 8 MEDLINE
AN 94178820 MEDLINE
TI Promotion of natural killer cell growth in vitro by bispecific (anti-CD3 x anti-CD16) antibodies.
AU Malygin A M; Somersalo K; Timonen T
CS Department of Pathology, University of Helsinki, Finland..
SO IMMUNOLOGY, (1994 Jan) 81 (1) 92-5.
Journal code: GH7. ISSN: 0019-2805.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9406
AB Bispecific heteroconjugated F(ab')2 fragments were prepared from pepsin-digested monoclonal OKT3 (anti-CD3) and 3G8 (anti-CD16) antibodies with 5,5'-dithiobis- (2-nitrobenzoic acid). When these bispecific antibodies (BsA) were added to peripheral blood lymphocyte (PBL) cultures with 100 U/ml human recombinant interleukin-2 (rIL-2), preferable growth of natural killer cells occurred. After 3 weeks the frequencies of CD56+ and CD56+3- cells in cultures with BsA were 74 +/- 7% and 65 +/- 7%, respectively, compared with 48 +/- 6% and 29 +/- 7% in control cultures. The frequencies of CD3+ lymphocytes in the presence of BsA, cells from 1-day cultures were labelled with fluorescein isothiocyanate (FITC)-conjugated anti-CD3, CD4 and CD8 monoclonal antibodies (mAb) and propidium iodide which stains dead cells. Flow cytometry revealed that more than 95% of the dead cells in cultures with BsA were CD3+. Thirty-seven per cent of CD3+, 43% of CD4+ and 17% of CD8+ cells were dead on day 1, and after 3 days the CD4+/CD8+ ratio among viable lymphocytes was 1.6 in the control and 0.5 in BsA

cultures. Taken together, these results show that bispecific (anti-CD3 x anti-CD16) F(ab')2 fragments are strongly immunomodulatory by inducing the killing of T cells by CD16+ cells.

L232 ANSWER 6 OF 8 MEDLINE DUPLICATE 2
AN 94348765 MEDLINE
TI Production and use of anti-FcR bispecific antibodies.
AU Fanger M W; Graziano R F; Guyre P M
CS Department of Microbiology, Dartmouth Medical School, Lebanon, New Hampshire 03756..
SO IMMUNOMETHODS, (1994 Feb) 4 (1) 72-81. Ref: 91
Journal code: B3R. ISSN: 1058-6687.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 9412
AB Bispecific antibodies (BsAb) are antibodies with two different specificities. BsAb composed of anti-Fc gamma R Ab linked to anti-target Ab have been useful in exploring the function of the three classes of human Fc gamma R. In addition, BsAb have been developed as new agents for immunotherapy which can join together different molecules or cells. In directed or redirected cytotoxicity, BsAb that bind both to target cells (pathogens or tumors) and to triggering molecules on leukocytes such as Fc gamma R are used to focus normal cellular immune defense mechanisms specifically to the tumor cell or infectious agent. Limited clinical trials have demonstrated little toxicity and promising responses. This ability to redirect normal cytotoxic mechanisms to kill tumors, infectious agents, or infected cells makes BsAb powerful new therapeutic tools. In addition, BsAb are being used to target other appropriate molecules to Fc gamma R, including antigens as vaccine adjuvants and immune complexes. This review focuses on BsAb in which one specificity is directed to Fc gamma R on human leukocytes. It considers applications of these reagents and discusses the progress toward an understanding of the construction and use of BsAb in therapy.

L232 ANSWER 7 OF 8 MEDLINE DUPLICATE 3
AN 94064175 MEDLINE
TI A CD16/CD30 bispecific monoclonal antibody induces lysis of Hodgkin's cells by unstimulated natural killer cells in vitro and in vivo.
AU Hombach A; Jung W; Pohl C; Renner C; Sahin U; Schmits R; Wolf J; Kapp U; Diehl V; Pfreundschuh M
CS Medizinische Klinik, Universitat des Saarlandes, Homburg/Saar, Germany..
SO INTERNATIONAL JOURNAL OF CANCER, (1993 Nov 11) 55 (5) 830-6.
Journal code: GQU. ISSN: 0020-7136.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)

LA English
FS Priority Journals; Cancer Journals
EM 9403
AB In order to target NK cells against the Hodgkin's-derived cell line L540, we developed bispecific monoclonal antibodies (Bi-MAbs) by somatic hybridization of the 2 mouse hybridoma cell line HRS-3 and A9 which produce monoclonal antibodies (MAbs) with reactivity against the Hodgkin and Reed-Sternberg cell-associated CD30 antigen and the CD16 antigen (Fc gamma III receptor), respectively. The CD16 MAb-producing cell line A9 was selected as a partner for HRS-3 because of its efficiency in inducing lysis of the A9 hybridoma cells by resting NK cells. The hybrid hybridoma cell line HRS-3/A9 produced the supernatant with the strongest bispecific reactivity and was repeatedly subcloned and used for ascites production. Crude supernatant and purified HRS-3/A9 Bi-MAb triggered specific lysis of the CD30+ Hodgkin's-derived cell line L540, but not of the CD30- cell line HPB-ALL by unstimulated peripheral-blood lymphocytes and NK-cell-enriched populations. Moreover, treatment of SCID mice bearing heterotransplanted human Hodgkin's tumors with HRS-3/A9 and human peripheral blood lymphocytes induced specific complete tumor regression in 10/10 animals. We thus report successful tumor treatment in an in vivo model using NK-cell-associated Bi-MAbs and show that the Bi-MAb HRS-3/A9 is an efficient promoter of the anti-tumor effects of NK cells in vitro and in vivo.

L232 ANSWER 8 OF 8 MEDLINE
AN 94006336 MEDLINE
TI Use of anti-CD3 and anti-CD16 bispecific monoclonal antibodies for the targeting of T and NK cells against tumor cells.
AU Ferrini S; Cambiaggi A; Sforzini S; Canevari S; Mezzanzanica D; Colnaghi M I; Moretta L
CS Istituto Nazionale per la Ricerca sul Cancro, Genova, Italy..
SO CANCER DETECTION AND PREVENTION, (1993) 17 (2) 295-300.
Journal code: CNZ. ISSN: 0361-090X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9401
AB To target T lymphocytes against EGF-R+ tumors, we constructed anti-CD3/anti-EGF-R bimAbs either by the generation of a hybrid hybridoma (quadroma) or by a chemical cross-linking method. Analysis of the in vitro functional activity of these two different constructs indicated that the quadroma-secreted bimAb was more efficient in targeting the CD3+8+ clones against EGF-R+ target cells with respect to the bimAb produced by chemical method. In addition, the quadroma-produced bimAb is able to induce cytolysis of EGF-R+ tumor cell lines of PHA-induced lymphoblasts that had been expanded in IL-2-containing medium, whereas tumor cells lacking expression of EGF-R were not lysed. Resting PBL targeted by the bimAb did not display significant cytotoxicity against the relevant tumor. An anti-CD16 hybridoma (IgG1) was fused with an anti-folate-binding protein hybrid (IgG2a) to construct bimAbs to target NK cells

against NK-resistant ovarian carcinomas. The hybrid IgG1/IgG2a bimAb triggered the specific lysis of relevant target cells by resting NK cells, but it was ineffective when CD8+TCR alpha/beta+ cultured cell populations were used as effectors. Only marginal increases of cytolytic activity could be induced by the bimAb when IL-2-activated PBL (i.e., LAK cells) were used as effectors due to the high cytolytic activity of these cells against the relevant tumors in the absence of bimAb. The possible use of anti-CD16 or anti-CD3 bimAbs for the development of different cellular immunotherapy strategies against cancer is discussed.

FILE 'HOME' ENTERED AT 16:31:42 ON 02 APR 96

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S9         65   S6 OR S7 OR S8
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S11         2   "BISPECIFIC ANTIBODY 2B 1"
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           5536  S13
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E12        1       FC GAMMA 2B RECEPTOR

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E18        1       FC R LYMPHOCYTES
E19        1       FC REACTING FACTOR
E20        1       FC REACTIVE FACTOR
E21     5828   13  FC RECEPTOR
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E23        1       FC RECEPTOR --DRUG DEVELOPMENT --DV
E24        1       FC RECEPTOR --DRUG THERAPY --DT

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E24        1        BISPECIFIC ROSETTE FORMING CELLS

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E5         1        BISPECIFIC CELLS
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E7         1        BISPECIFIC MONOCLONAL ANTIBODY
E8         1        BISPECIFIC ROSETTE FORMING CELLS
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E12        4        BISPECIFICS

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     S12   663  BISPECIFIC(W)ANTIBODY?
?e cd16


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              18  S7
              20  S8
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E7         4  BISPECIFICS
E8         2  BISPECIFIQUE
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E12        3      BISPECIFIC ANTIBODY --DRUG INTERACTION --IT

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               27  AU=RING D B
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E9       124  AU=RING E
E10        2  AU=RING E D
E11        1  AU=RING E F
E12       13  AU=RING E F J

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?e au=ring d.?


Ref    Items  Index-term
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E9         3  AU=RING DL
E10        1  AU=RING DM
E11        1  AU=RING DR
E12        3  AU=RING DS

          Enter P or PAGE for more
?s e4

      S2      18  AU="RING D.B."
?e au=ring d b


Ref    Items  Index-term
E1         7  AU=RING CS
E2        58  AU=RING D
E3        27 *AU=RING D B
E4         2  AU=RING D F
E5         1  AU=RING D M
E6        18  AU=RING D R
E7         1  AU=RING D S
E8        24  AU=RING D.
E9        18  AU=RING D.B.
E10        4  AU=RING D.L.
E11        1  AU=RING D.M.
```

```
Ref    Items  Index-term
E1         4  CERBARA
E2         1  CERBA1
E3        51 *CERBB
E4         1  CERBB-2
E5         1  CERBBROSPINAL
E6         1  CERBB1
E7        15  CERBB2
E8         3  CERBB3
E9         9  CERBC
E10        1  CERBEAU
E11        1  CERBEBR
E12        2  CERBEBRAL

          Enter P or PAGE for more
?e her


Ref    Items   RT  Index-term
E1        16       HEQ2
E2        16       HEQ2NEQ2
E3     62389      *HER
E4         1       HER CONTACTS
E5         1       HER DEAREST FRIEND
E6         0    1  HER 2 ONCOGENE
E7         4       HER 25
E8         1       HER-NEU
E9        11       HER-1
E10        5       HER-1 PROTEIN
E11        1       HER-1/HER-2
E12       56       HER-2

          Enter P or PAGE for more
?e e6


Ref    Items Type  RT  Index-term
R1         0        1 *HER 2 ONCOGENE
R2       173   P    5  ONCOGENE NEU
?s r2

     S18     173  "ONCOGENE NEU"
?s s18 or her(w)2? or her(w)neu? or cerbb? or c(w)erb(w)b? or cerb(w)b? or
p185?

>>>File 155 processing for 2? stopped at  2NAPQI
>>>File 155 processing for NEU? stopped at  NEUROCHIRURGIEN
>>>File 155 processing for B? stopped at  BACTERIOSIS
>>>File 155 processing for B? stopped at  BACTERIOSIS
Processing


INT CONF
!
?s s18 or her(w)2? or her(w)neu? or cerbb2 or cerbb(w)2 or c(w)erbb or
c(w)erbb(w)2 or cerbb2 or p185?
```

```
>>>"S18" does not exist
>>>File 155 processing for 2? stopped at  2NAPQI
>>>File 155 processing for NEU? stopped at  NEUROCHIRURGIEN
Processing


INT CONF
!
?ds


Set      Items   Description
S1          20   AU="RING DB"
S2          18   AU="RING D.B."
S3          27   AU="RING D B"
S4          92   AU="RING CS" OR AU="RING D" OR AU="RING D B"
?LOGOFF

       11oct96 08:09:08 User231815 Session A21.2
            $4.50    0.150 Hrs File155
     $4.50  Estimated cost File155
            $3.30    0.055 Hrs File55
     $3.30  Estimated cost File55
            $3.96    0.044 Hrs File73
     $3.96  Estimated cost File73
            OneSearch, 3 files,  0.250 Hrs FileOS
    $11.76  Estimated cost this search
    $11.76  Estimated total session cost   0.256 Hrs.
Logoff: level 42.09.02 A  08:09:09


CLR DTE 82
```